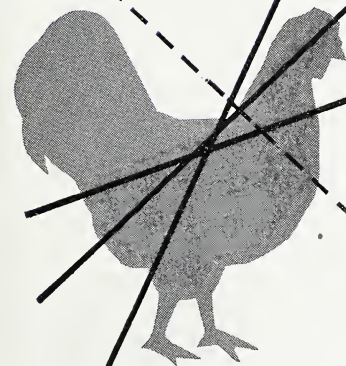
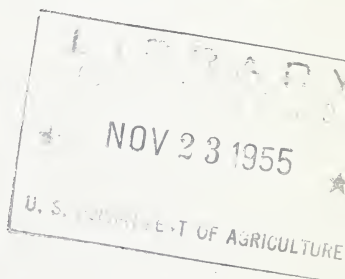
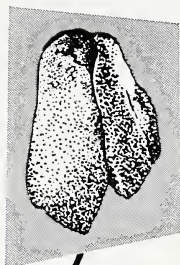


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LYMPHOMATOSIS IN CHICKENS



Circular No. 970

U. S. DEPARTMENT OF AGRICULTURE

The U. S. Regional Poultry Research Laboratory, East Lansing, Mich., was established to conduct research on the improvement of viability in poultry. Plans for this laboratory were approved by the Secretary of Agriculture on December 23, 1937.

The establishment of this laboratory was sponsored by the agricultural experiment stations of the States of Connecticut, Delaware, Illinois, Indiana, Iowa, Kansas, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, Nebraska, New Hampshire, New Jersey, New York, North Dakota, Ohio, Oklahoma, Pennsylvania, Rhode Island, South Dakota, Vermont, West Virginia, and Wisconsin.

Because of the high mortality from lymphomatosis among chickens, studies were initiated at this laboratory and at certain cooperating experiment stations to determine the cause of lymphomatosis and to develop measures for its prevention and control.

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LYMPHOMATOSIS IN CHICKENS¹

Prepared under the direction of the Animal and Poultry Husbandry Research Branch, in cooperation with the Animal Disease and Parasite Research Branch, Agricultural Research Service.

INTRODUCTION

Lymphomatosis is the most baffling and destructive disease of chickens in the United States. In addition to the many millions of chickens that die each year with typical clinical and post mortem findings, millions of others die without showing specific evidence of a known infectious disease. Such deaths are frequently classified as being from undetermined or nonspecific causes, but they may be a result of latent infections of lymphomatosis. This conclusion is based upon the observations that deaths from undetermined causes often parallel deaths from lymphomatosis, and that a large proportion of the chickens in infected flocks, although they appear healthy, are actually infected.

The purpose of this circular is to summarize the results of research to date and to provide as much helpful information as possible for various groups who must deal with the problem, including farmers, poultrymen, county agents, poultry improvement employees, vocational agriculture teachers, veterinarians, and experiment station workers.

DISTRIBUTION AND ECONOMIC IMPORTANCE

Lymphomatosis, in its various forms, is distributed widely in flocks of chickens throughout the United States, and has been reported in all parts of the world in which chickens are raised. Unfortunately, no statistical data on the incidence of different forms of lymphomatosis are compiled in poultry diagnostic laboratories in this country.

The mortality of adult chickens continues to be a limiting factor in the economical production of chickens and eggs. The importance of this factor was shown by the results obtained at the New York Random Sample Test (28)² where 26 advanced poultrymen from different sections of the country entered their stock for the 3 years 1949 to 1952. The yearly average total mortality of the hens hatched as chicks at this test was 35.3 percent. Lymphomatosis was responsible for 14.5 percent of this loss, two-thirds of which was caused by the visceral form of the disease.

It is estimated that if the mortality from lymphomatosis among chickens in the United States was only 14.5 percent in 1953, more than 53 million mature chickens, with a total value of at least \$73 million,

¹ This publication was prepared at the U. S. Regional Poultry Research Laboratory, East Lansing, Mich., by the laboratory staff: Berley Winton, director; B. R. Burmester, biologist and veterinarian; E. M. Denington, medical technologist; A. M. Lucas, cytopathologist; S. W. Leshner, embryologist (resigned), and Nelson F. Waters, geneticist.

² Italic numbers in parentheses refer to Literature Cited, p. 15.

died from this disease. This staggering loss does not take into account the less tangible losses caused by an impairment of growth in young stock, a reduction in egg production of laying birds, and losses from failure to use housing facilities to capacity.

EARLY HISTORY OF LYMPHOMATOSIS IN THE UNITED STATES

In 1905, Butterfield (15) reported a disease condition of chickens in the District of Columbia and in Michigan which would appear, from the description given, to be what we identify now as visceral lymphomatosis. A report in 1920 (unpublished) by Goodale of the Poultry Department of the Massachusetts Experiment Station, indicates that "fowl paralysis," or neural lymphomatosis, had been prevalent in the station flock since 1913, and probably since 1911. The 25-percent mortality in this flock in 1918-19 was from a disease characterized by extreme emaciation, possibly due to paralysis of the digestive tract. Blindness (perhaps caused by ocular lymphomatosis) was also given in the 1920 report as having occurred among chickens at the Massachusetts station.

In 1921 Kaupp (27) reported that 15 cases of paralysis had been studied since 1914 in 7 States—Connecticut, New Hampshire, New Jersey, North Carolina, Maryland, Massachusetts, and Virginia. Between 1920 and 1930 the various forms of lymphomatosis appeared among chickens on most farms in practically every section of the United States. High mortality from the different forms of lymphomatosis continued almost unabated during the next decade, after which it appeared to be somewhat less. The general impression prevails among poultry producers that neural lymphomatosis caused its highest mortality prior to 1930. Thereafter, the visceral form became much more prevalent, and it continues to be responsible for two-thirds to three-fourths of all mortality caused by lymphomatosis. At no time has ocular lymphomatosis caused so high a death rate for the country as a whole as the other two forms.

MANIFESTATIONS OF LYMPHOMATOSIS³

The three common forms of lymphomatosis that are of great economic importance to the poultry industry are termed visceral lymphomatosis (big liver disease), neural lymphomatosis (fowl or range paralysis), and ocular lymphomatosis (iritis)—a terminology obviously derived from the organs affected and without reference to the cause.

During a 13-year period the mortality from naturally occurring lymphomatosis in chickens at the laboratory was classified as 73 percent visceral, 24 percent neural, and 3 percent ocular (37). In flocks where all forms of lymphomatosis were present, neural lymphomatosis usually appeared first, followed by the visceral, and then the ocular.

Lymphomatosis is limited primarily to chickens (20) although it has been reported in turkeys (1) and in pheasants (25). Typical

³ For a discussion of terminology in connection with the avian leukosis complex, see "Diseases of Poultry," 3d edition, pp. 453-507, edited by Biester and Schwarte, Iowa State College Press, 1952.

symptoms of paralysis have been observed in ducks (18), but as yet no lymphoid tumors have been reported.

Visceral Lymphomatosis

The most common manifestation of visceral lymphomatosis in chickens is the formation of tumors in the visceral organs (fig. 1). One or more organs may be affected. The lesions involved in enlargement of the liver, spleen, ovaries, and other organs of the body are caused by a multiplication of neoplastic or cancerous lymphoid cells. After this lymphomatous growth is activated, there is no known method of arresting it, and death eventually results.

Visceral lymphomatosis may be either acute or chronic. Fast growing pullets may become droopy and succumb within a few days; laying hens may cease laying abruptly, as shown by trap-nest records, and die within a short time. In contrast to this, other birds may be droopy in appearance for a relatively long time, become emaciated, and die after a long illness. Advanced cases may be detected before death by palpation of the enlarged liver. In fact, this condition is so typical that the term "big liver disease" has been used by poultrymen to describe it.

Neural Lymphomatosis

This form of lymphomatosis among chickens affects the nerves and often results in paralysis. Clinical manifestations are a drooping of one or both wings (fig. 2) and a weakness and lack of coordination of the legs which makes standing difficult and usually leads to inability to stand or walk. The affected chicken may lie on its side (fig. 3) with one leg extended forward and the other backward. Such a bird has difficulty in reaching feed and water and, furthermore, it is likely to be cannibalized by its penmates. If affected birds are identified soon after the onset of the disease, and are then isolated and provided with nourishment, they usually survive for some time, and occasionally make a partial, if not complete, recovery. Other less frequent symptoms are irregular movement or tilting of the head accompanied by difficult breathing.

Gross examination of chickens with neural lymphomatosis, particularly those in advanced stages, may show irregular or uniform enlargement with or without a yellowish discoloration of one or more of the nerves leading from the spinal column to one or both legs, to one or both wings (fig. 4), or to the neck. The nerves are swollen through an accumulation of lymphoid cells. Only rarely does this enlargement prove to be neoplastic.

The usual procedure at necropsy is to examine the brachial nerves leading to the wings, the sciatic nerves leading to the legs, the vagus nerves in the neck, and the nerves supplying the viscera. An enlargement of the larger nerves can be detected readily, but abnormalities of smaller nerves, particularly of the branches leading to the viscera, are more difficult to recognize. The cross striations of nerves, which are normal in healthy birds, may disappear with this disease. It is not always possible to find such changes at post mortem examination although typical clinical symptoms have been observed.

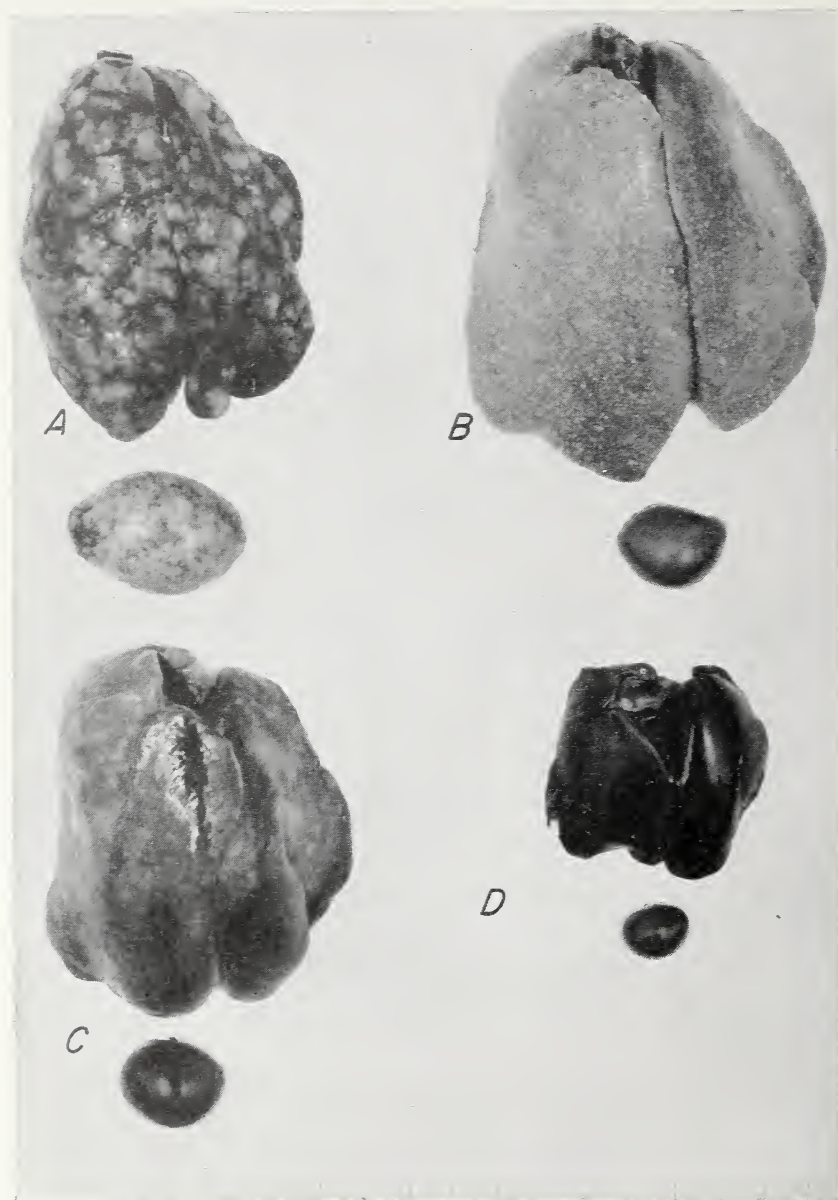


FIGURE 1.—*A*, Enlarged tumorous liver and spleen taken from a bird with visceral lymphomatosis. The lesions (light areas) in these organs are large. *B*, Enlarged liver and spleen with small lesions. *C*, Enlarged liver and spleen; the minute individual lesions cannot be seen with the naked eye. *D*, A normal liver and spleen. (Reduced in same proportion as inch scale below.)



FIGURE 2.—A bird with neural lymphomatosis, manifested by droopy wing.



FIGURE 3.—This chicken's posture is typical of the neural form of lymphomatosis characterized by paralysis of one or both legs.

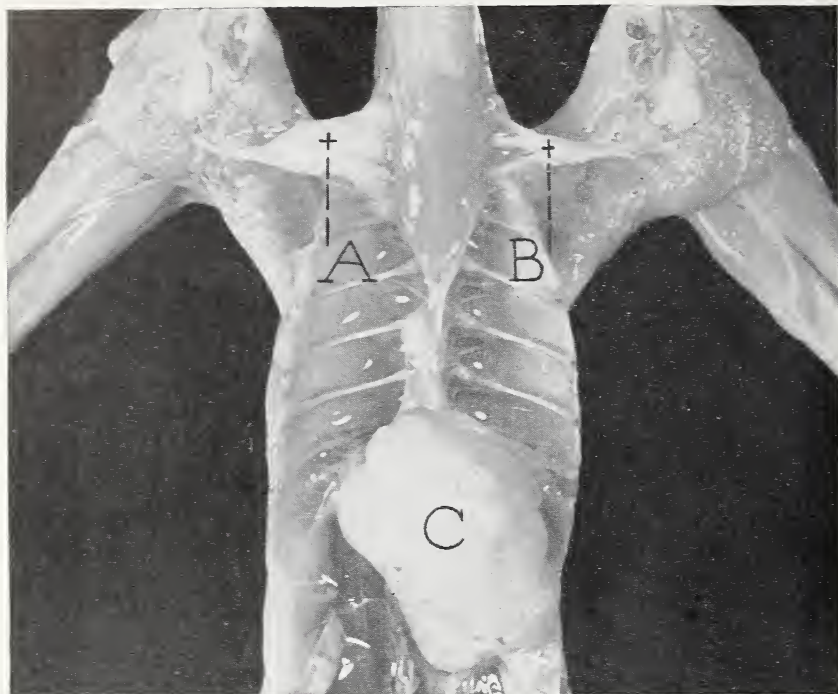


FIGURE 4.—Enlarged brachial plexus nerves (*A* and *B*) leading to the wings, indicate paralysis or neural lymphomatosis. The condition is more pronounced in the right wing (*A*) than in the left wing of this chicken. Visceral lymphomatosis is also clearly shown by the ovarian tumor (*C*).

Ocular Lymphomatosis

Ocular lymphomatosis results in impairment of the vision or complete blindness in one or both eyes (31). There is usually an alteration in the size and shape of the pupil of the eye (fig. 5), and in the color of the iris of the eye from an orange or reddish bay, in most breeds and varieties of chickens, to a gray appearance, because the normal pigmented iris has been invaded by lymphoid cells. The pupillary changes, such as clefts or irregular contour, and stoppage of movement such as that which takes place in accommodating to light changes or at death when the normal eye dilates, are regarded as being more diagnostic of ocular lymphomatosis than is the fading color in the iris, since in some breeds and crosses the iris is naturally gray; also, the color is influenced by the amount of yellow pigment in the diet and by egg production (2, 30).

ASSOCIATED DISEASES

Osteopetrosis and fowl leukemia are frequently classified with the different forms of lymphomatosis under the broad term, "avian leukosis complex," but further research is necessary to determine their relationship definitely.



FIGURE 5.—A bird with ocular lymphomatosis.

Osteopetrosis

An enlargement of the leg bones (fig. 6) is usually the first clinical symptom observed in chickens with osteopetrosis (26). As this disease, commonly called "big bone disease," progresses, other bones of the body become involved. In advanced cases, the affected chicken becomes stiff and has greater difficulty in reaching feed and water. A histological study of osteopetrosis shows that cells of the bone coverings which produce the bone are involved.

Although osteopetrosis has been reported in different sections of the United States, its occurrence ranges from an occasional bird in some flocks to a significant number in other flocks. The incidence of this disease is not high enough in the aggregate to be of major economic importance at this time.

At the Regional Poultry Research Laboratory not only a high incidence of visceral lymphomatosis but also a variable amount of osteopetrosis has occurred in young chicks inoculated with a particular strain of inoculum (8) from a chicken which apparently had only visceral lymphomatosis.

Fowl Leukemia

Fowl leukemia was reported in the United States (32) in 1914. However, its occurrence to date in flocks of chickens has not been sufficiently high to be of great concern economically to poultry producers. Nevertheless, the potentialities of leukemia are such as to

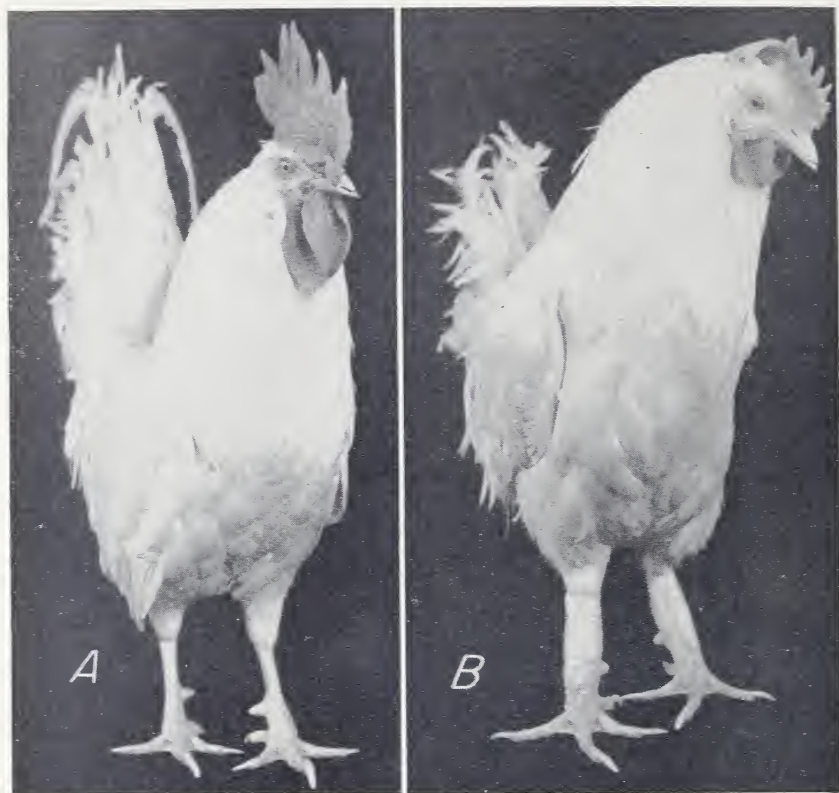


FIGURE 6.—Two chickens of the same age. *A*, Cockerel with leg bones of natural size. *B*, Cockerel showing enlargement of leg bones, and posture indicative of diseased condition.

warrant constant surveillance, especially since an examination of blood from the living bird is required for final diagnosis.

Leukemia is a neoplastic condition of the blood which results from excessive multiplication of either the immature red or white blood cells, or both. Chickens affected with this disease often develop a light yellow color about the head and an unthrifty appearance. The liver and spleen are usually enlarged and have a gray or a bright red color, depending on the cell type involved. This disease may be reproduced readily in young chickens by inoculation with filtrates or blood obtained from diseased birds. It is concluded by most authorities that fowl leukemia is caused by a virus.

THE CAUSATIVE AGENT OR AGENTS

The widespread dissemination of lymphomatosis in the United States soon after the different forms were observed created much interest and speculation as to the exact cause of this disease. This rapid and simultaneous spread implied that the disease was contagious and was caused by an infectious agent. The occurrence of neural lymphomatosis on one farm, ocular on a second, visceral on a

third, and perhaps a combination of two forms of lymphomatosis (fig. 4) on some or all of the farms, added perplexity regarding the causative agent. A high incidence of the complex in a flock one year and almost complete absence of it the subsequent year, without any apparent change in hatching, brooding, and maintenance procedures, gave rise to further questions about its cause. Inability of investigators to associate the causative agent positively with the diet likewise emphasized the difficulties in determining the cause of lymphomatosis. Rarely has either one form or a combination of forms reached the epizootic proportions that are common with more highly contagious diseases such as fowl pox, Newcastle disease, and infectious bronchitis.

Experiments show that visceral lymphomatosis usually can be reproduced by inoculating young chicks with cellular material from diseased birds. Furthermore, more recent research (3, 8, and 19) demonstrates that this form of the disease may result from the use of inoculating material that has been centrifuged or passed through a filter capable of removing all bacteria and particles of tissue.

The causative agent in such filtrates (5) may be kept viable for several hundreds of days at extremely low temperatures, or it may be made inactive by temperatures of 125° to 135° F., by formaldehyde, or by ultraviolet irradiation. These biological and physical properties of the agent support the contention that visceral lymphomatosis, in particular, is caused by a virus. Evidently this virus is different from the agent or agents responsible for neural and ocular forms of the disease (9, 37). It also appears to be different from the virus that causes osteopetrosis.

The concurrent association of lymphomatosis with other diseases of chickens has led many producers to believe that other diseases are a predisposing factor to lymphomatosis. Although research workers have demonstrated that visceral lymphomatosis is caused by a virus and that the presence of other pathogens is not necessary for its expression, coccidia or other parasites may be the mechanical means of spreading the virus.

SPREAD OF LYMPHOMATOSIS

How the different forms of lymphomatosis are spread is of much concern to farmers, poultry breeders, hatcherymen, and research workers. The elusiveness of the causative agent and the insidious nature of the disease combine to make investigations of its spread more difficult.

Visceral Lymphomatosis

Studies of transmission (9, 10, 17, 33) have definitely shown that visceral lymphomatosis is spread by contact and through hatching eggs. Recent investigations (11) indicate that washings from the oral and nasal passages of chickens with visceral lymphomatosis contain the virus of this disease. Furthermore, the feces from certain chickens contain the virus. Therefore, virus from either of these sources may serve to infect the environment.

Other studies (12) of transmission by inoculation reveal that visceral lymphomatosis may be spread through such natural routes as the nasal passages, the oral cavity, or the cloaca. In other words, any

mucous membrane normally exposed to the external environment may serve as the avenue of entry for the virus of visceral lymphomatosis.

Although circumstantial evidence supporting the theory of egg transmission of lymphomatosis has been accumulated over the years by many investigators, it was not until recently that direct proof was obtained (10, 17). Hatching eggs from infected hens whose sibs and progeny had died in large numbers from lymphomatosis were used to produce 15-, 18-, and 21-day-old embryos from which inoculating material was procured. These inoculums were injected into young chicks (usually 1 day of age) from laboratory stock with a relatively low incidence of lymphomatosis. The recipient chicks and control penmates were brooded and maintained under rigid quarantine and isolation for 270 to 300 days.

Analyses of data involving more than 2,500 chicks showed that the infectious agent of visceral lymphomatosis was present in a significant number of chick embryos. The rate of mortality from visceral lymphomatosis in one lot of inoculated chicks was as high as 88 percent, and the average of 29 percent for all inoculated lots definitely established hatching eggs as a source of the infective virus. Other forms of lymphomatosis are not necessarily spread in the same manner, nor are they necessarily caused by the same virus. A large number of the chickens used to produce hatching eggs were normal in appearance but shed the virus of visceral lymphomatosis in their eggs. These results warranted placing all these birds in the category of "carrier" hens.

The progeny of carrier hens may not develop a high incidence of lymphomatosis. On the average, the incidence of lymphomatosis in the progeny of carrier hens was not significantly greater than that in the progeny of noncarrier hens (14). However, the chicks of carrier hens are an important source of infection for chicks of noninfected stock. When an extract of dust, down, and other debris collected from an incubator containing chicks of an infected flock was injected into noninfected susceptible chicks, it caused a high incidence of visceral lymphomatosis. Furthermore, when chicks of an infected flock were hatched and brooded with chicks of a noninfected flock, the latter developed a high incidence of visceral lymphomatosis, whereas the former did not. When hatched together but brooded separately, the chicks from the two flocks had about the same incidence of the disease, and thus the brooding period appears to be more important than the hatching period in the transmission of visceral lymphomatosis.

Egg transmission of the disease from chicks hatched from infected eggs to chicks from eggs of hens that have had no experience with the virus is of more importance than transmission of the disease from infected hens to their own chicks.

The fact that visceral lymphomatosis is spread by contact from diseased bird to healthy chicken after hatching, and also from infected hen to chick through the egg, establishes it as a contagious disease. It is the only known neoplastic, malignant disease that is spread by contact, although the leukemias and the sarcomas of the Rous type are likewise caused by highly infectious viruses. This feature more than any other (10) makes visceral lymphomatosis a unique disease, as well as the most destructive disease of chickens in the United States. Furthermore, transmission through the egg pro-

vides a chain of infection which cannot be broken so easily as that of other virus diseases which are not egg borne, or in which the virus is shed for only a short time.

Neural Lymphomatosis

It is estimated that approximately 20 percent of all deaths from lymphomatosis in the United States are from the neural form of the disease. It is usually the first form of the disease to be observed clinically, often causing paralysis in young chickens. Paralysis is also observed in older chickens.

Investigations (21, 36) indicate that neural lymphomatosis is spread by contact during the early brooding period. In some experiments degree of proximity between chicks and adult chickens has influenced greatly the incidence of this form of the disease. However, the specific means of transmission was not determined in these tests. There is no evidence (10, 17) that neural lymphomatosis is spread from parent to chick through the hatching egg.

Ocular Lymphomatosis

The incidence of ocular lymphomatosis at this laboratory has been so low that there has been no opportunity to study intensively its transmission. However, it occurs regularly in 2 to 3 percent of the chickens year after year, but it has not occurred among those used in studies of transmission by inoculation.

FACTORS AFFECTING RESISTANCE OF CHICKENS TO LYMPHOMATOSIS

Results of experiments (6, 7, 22, 34, 35) show that genetic resistance, virus-stimulated resistance (resistance due to antibodies), and age of chicken at time of exposure are important factors influencing the amount of lymphomatosis in any flock.

Selective breeding at the Regional Poultry Research Laboratory (34) has resulted in the development of one line of White Leghorns in which there is only about one-fourth as much mortality from lymphomatosis as in a second line, when the two lines are raised together. Hutt and Cole (23) at Cornell University developed a resistant line that has shown a yearly mortality from lymphomatosis of 5 percent or less, as compared with a mortality of 50 to 65 percent in their susceptible line, both lines being given similar exposure.

In recent experiments Burnester (6) has shown that the resistance of chickens to visceral lymphomatosis can be greatly increased by giving the dams a series of injections containing the virus. The injected virus stimulates the formation of neutralizing antibodies in the hens (7) and results in a partial immunity in the offspring. The incidence of the disease was low among chicks of the hens that were known to shed the virus into their eggs. It is likely that carrier hens not only shed virus into their eggs, but also antibodies, which give some measure of protection to the progeny.

The younger the chicks at the time of exposure to the virus of lymphomatosis, either under natural conditions (39) or by inoculation

(5), the greater the incidence of the disease. When day-old isolated chicks that were relatively free of the infection were mixed with day-old infected chicks, 38 percent of the former lot developed lymphomatosis by the time they were 500 days of age. When chicks from the same parental stock were held in isolation for 30 days and then mixed with infected chicks of the same age, the mortality was only 15 percent.

In another experiment (5) chicks were inoculated at 2, 16, 30, 58, and 114 days of age. When the chickens were 300 days of age, the incidences of visceral lymphomatosis were 95, 88, 47, 53, and 31 percent respectively. Thus, age at time of exposure is an important factor in determining the response.

INFLUENCE OF AMOUNT OF EXPOSURE ON INCIDENCE OF LYMPHOMATOSIS

By isolation and quarantine procedures, Waters (40) has maintained a group of susceptible chickens with a minimum of lymphomatosis. Cole and Hutt (16) reported a marked reduction in lymphomatosis when chickens were raised in isolation. When chicks were reared in close proximity to adult stock, or were hatched and brooded with infected stock of the same age (14, 35), a marked increase in lymphomatosis resulted.

In studies on the effect of brooding and raising noninoculated susceptible chickens with their inoculated sibs and penmates (9), five lots of chicks were brooded in cubicles made of plywood to furnish limited isolation. The cubicles varied in size but provided a comparable amount of floor space for each chick. Both inoculated and noninoculated chicks of the same age and hatch, and from the same parental stock, were brooded in the cubicles for the first 90-day period. They were then transferred to a large pen where they were maintained together until they were 300 days of age. This test showed that the larger the proportion of chicks inoculated, the greater was the incidence of visceral lymphomatosis among the noninoculated penmates.

In research (13) on the relation between response of susceptible chickens and the dose of virus of visceral lymphomatosis injected intraperitoneally, an intermediate increase in dosage rate caused a corresponding increase in percentage of lymphomatosis, but when the dosage was increased tenfold, the percentage of lymphomatosis did not increase in proportion. In one experiment where the highest dose of inoculum was 100,000 times the lowest dose, there was a difference of only 26.7 percent in the occurrence of visceral lymphomatosis. However, the average age at death decreased rapidly with the increase in dose.

AGE OF CHICKENS WHEN AFFECTED WITH LYMPHOMATOSIS

Although young chicks become infected more readily than older chickens when exposed to lymphomatosis, age is no indication as to when the disease may be manifested. It is not known what factors provide optimum conditions for activation of the causative agent in the affected bird.

The average age at death from visceral lymphomatosis among the laboratory birds was 322 days, and for those with neural involvement

241 days. Mortality from either the visceral or the neural form of the disease may start as early as 30 days of age and continue in a particular population of chickens as long as any of them remain alive, even to 1,800 days of age or older (37, 38).

CONTROL MEASURES FOR LYMPHOMATOSIS

No one of the various forms of lymphomatosis can be prevented by presently known methods of vaccination or by the use of other prophylactic measures. No drugs or other therapeutic treatments have been found to cure this disease complex. Chickens affected with any form of lymphomatosis rarely show complete or partial recovery. Dietary factors are not known to be associated with its course.

Selective Breeding

Various investigators have found it possible, through selective breeding, to develop and maintain chickens that will have an inherent resistance to lymphomatosis (22, 23, 24, 29, 34). Selective breeding, to be most effective, necessitates a knowledge of genetics, pedigree breeding, and familiarity with the mortality pattern of the different families (progeny of each male and female) of chickens.

If this breeding procedure is followed for a number of years it will result in the development of lines of birds that have a relatively low mortality from this disease, assuming that the disease is present and that the chickens used for breeding purposes are exposed to the causative agent.

When a selective breeding program is accompanied by prompt removal from the premises of all chickens that have lymphoid tumors or that show definite clinical symptoms as soon as they are detected, infection of the premises will eventually be reduced and exposure of chicks to the causative agent will be limited. Under these circumstances, a low incidence of lymphomatosis may be the result of little or no exposure rather than the result of increased inherent resistance. This conclusion is also suggested by the high incidence of lymphomatosis on the farms of the poultry breeder's customers, where there often is high exposure to the infection in contrast with the low rate of mortality on the breeder's home premises.

The possibility of using selective breeding as a control measure for lymphomatosis is illustrated by the research done at the Regional Laboratory during a 13-year period and at Cornell University over an 18-year period. At the Laboratory, White Leghorns (from line 6) that were selected for resistance to the disease had an average yearly mortality of 12 percent from lymphomatosis, whereas White Leghorns (in line 15) that were selected for susceptibility to the disease showed an annual loss of 44 percent. Both groups of chickens were hatched, brooded, and maintained together during the entire experimental period of 500 to 600 days, which indicates that the variation in mortality from lymphomatosis can be attributed to genetic influence.

The variation in mortality from lymphomatosis between susceptible and resistant strains of White Leghorns at Cornell University was even more marked than at the Regional Laboratory. Investigators there reported a mortality of 51 to 64.5 percent among their susceptible strains, which were hatched in 1948, 1949, and 1951, as compared with

a loss of only 2.7 to 7.8 percent among their resistant strains. The susceptible and the resistant strains of birds were brooded, raised, and maintained together for 500 days.

The predominant form of the disease among the chickens at the Regional Laboratory was visceral lymphomatosis; at Cornell University it was the neural form. Apparently the genetic response can be expected in breeding for resistance to lymphomatosis, irrespective of the fact that these two forms of the disease are caused by different agents and are spread by different means.

In following a selective breeding program, the poultry breeder must first identify the most desirable males and females in his flock, then make matings in preparation for the new hatching season. At this point in his breeding and management program, he must decide upon a specific procedure to follow in exposing a portion of the chicks to lymphomatosis, and in protecting the remaining chicks from the disease complex by raising them in isolation. In other words, exposure of prospective breeding stock or representative samples of this stock at a young age to the causative agent is essential for the identification and selection of the most resistant families.

Isolation

As previously mentioned, isolation of young chicks, especially during the brooding period, reduces exposure to the causative agent and affords some protection against lymphomatosis. Good brooding isolation can be provided by segregating chicks by source and by hatch, keeping them in sanitary quarters, and away from all other chickens, and having a caretaker who does not come in contact with other poultry. Most farmers cannot provide ideal environment but they should make every effort to provide optimum conditions as regards sanitation, quarantine, and isolation. Even when good isolation is provided, a moderate to high incidence of visceral lymphomatosis often occurs, the virus of the disease apparently being introduced through chicks from infected eggs, by infected excrement, or by other unknown means. For this reason it is highly advisable to hatch chicks from different flocks in different hatches or different hatchers, and to maintain the separation by raising the chicks in different compartments, pens, or houses.

Farmers whose chickens show a high mortality from lymphomatosis often raise the question as to the advisability of disposing of their flock and replacing it with birds from a different source, in the hope that the new stock would have greater genetic resistance to the disease complex. Whether such a practice is warranted depends on such factors as (1) the prevailing mortality, (2) the financial losses that will result from not having the houses and other facilities in use until other mature chickens are available, and (3) the ability to obtain stock that is actually highly resistant to lymphomatosis. Aside from information obtainable through official random sample tests and standard laying tests, a prospective purchaser must depend on the poultry breeder for information that will enable him to judge the relative merit of different strains of chickens, with respect to their resistance to lymphomatosis and to other economic qualities.

Farmers also frequently ask whether the age of parental stock has any relation to mortality from lymphomatosis of the progeny. At this time, there appears to be no advantage in using hens instead of pullets for breeding stock.

A third important question refers to the effect of crossbreeding (crossing of breeds, varieties, and lines within the same breed and variety) on the incidence of lymphomatosis in progeny. Although information available on this subject is limited, results from the crosses of inbred lines of White Leghorns made by Waters (38) indicate that hybridization does not decrease the mortality from lymphomatosis.

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